

**Conclusion:** The effects of fasting during Ramadan on stable patients with chronic heart failure are minimal. The majority of patients with stable cardiac disease can fast during Ramadan without significant detrimental effects.

## 0105

### Evaluation of management of heart failure and sleep apnea syndrome in a cardiac intensive care unit

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**Background:** Sleep apnea syndrome (SAS) is seen in approximately 40% of patients with congestive heart failure. SAS is also associated with an increased mortality in patients with systolic heart failure. The aim of this work was to evaluate the screening for SAS in patients hospitalized for acute congestive heart failure in a cardiac intensive care unit.

**Method:** We performed a single-center retrospective study of patients hospitalized for congestive heart failure with altered left ventricular ejection fraction (lvef)  $\leq 45\%$  from June to December 2013. We assessed the screening for sleep apnea syndrome using Epworth sleepiness scale.

**Results:** We included 51 patients. Mean values were for age 76 y. o, men 54%, body mass index 27 kg/m<sup>2</sup>, lvef 32%, diabetes 34%, hypertension 40%, NYHA III 38% and NYHA IV 50%. Only 14% (7) of patients were screened for SAS using the Epworth scale. All of them benefited a polysomnography at less one month after discharge. 6 of them (85%) were diagnosed with SAS.

**Conclusion:** Although Epworth scale is a useful and easy tool, Apnea syndrome remains underdiagnosed. Because of its high prevalence, poor outcome, and the beneficial effects of treatment, cardiologist should be more aware of SAS

## 0174

### Radiotherapy for Breast Cancer and early detection of Cardiotoxicity (REBECCA): a prospective cohort study

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Breast radiotherapy (RT) techniques used until the 1990s were responsible for increased mortality due to long term cardiac complications. Since the 2000s, improved techniques of RT have appeared leading to different dose distributions to organ at risks such as heart. But up to now, little is known on their cardiac toxicity. The aim of the REBECCA study is to evaluate whether helical tomotherapy (HT) induces cardiac toxicity that could be detected in the first two years after treatment based on analysis of sub-clinical functional and anatomical cardiac lesions at myocardial and coronary levels and evolution of circulating biomarkers. REBECCA study is a prospective cohort study that will include 120 women treated with adjuvant HT for breast cancer at the Institut Universitaire du Cancer Toulouse-OncoPole (IUCT), and followed up for 2 years after RT. Women aged 40 to 70 years, surgically treated at IUCT for breast cancer and for whom adjuvant

RT with HT is indicated, without chemotherapy will be eligible for the study. Follow-up will include measures of a panel circulating biomarkers, coronary plaque index based on Coronary computed tomography angiography and myocardial strain based on 2D-speckle tracking echocardiography. Absorbed doses will be evaluated for the whole heart and for each different parts of heart, in particular coronary arteries. Analysis will focus on dose-response relationship between subclinical cardiac lesions, biomarkers, and different organ absorbed doses. Furthermore, this study aims to create a bio-bank of plasma and blood of this cohort for future investigations. This clinical research study is a novel approach to early detect cardiotoxicity of current breast RT, combining anatomical and functional heart consequences based on cardiac imaging, a panel of circulating biomarkers and a detailed heart dosimetry. With this approach, REBECCA aims to improve understanding of the mechanisms and circumstances that underlie the development of potential heart side effects and sequelae.

## 0290

### Clinical, epidemiological and etiological characteristics of pulmonary hypertension and its prognostic value in chronic systolic heart failure: a report from the Ibn Rochd-HF Registry

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**Purpose:** Pulmonary hypertension (PH) is a predictor of mortality and morbidity in patients with chronic heart failure (HF) but it is poorly described in our population. We sought to study, the prevalence, determinants, and prognostic significance of PH in a large representative population with HF.

**Methods:** We retrospectively studied 1613 patients with HF. Systolic pulmonary artery pressure PASP was determined by echocardiography, PH was defined as PASP > 50 mmHg.

**Results:** The proportion of patients with PASP > 50 mm Hg was: 12,15% (196/1613), with an average age of 69 years, there was 40,30% women and 59,70% men, 96,43% of patients had low LVEF (left ventricular ejection fraction) and only 1,02% had preserved LVEF. The ischemic etiology of HF was predominant (17,86%). Most patients were receiving diuretics (66,33%), beta-blockers (58,16%) and ACE inhibitor (86,22%), whereas 55, 10% were on Spironolactone. The etiology of PH was mostly due to the left heart disease, while 1.02% was due to primary HP and 2.04% patient had COPD (Chronic obstructive pulmonary disease). Patients with PH had a rate of 6.06% of AHFD (acute heart failure decompensation) occurrence. We note that patients with both PH and RV dysfunction had a greater risk of AHFD (14,28%).

**Conclusion:** PH is common in HF patients, associated with worse LV function and provide incremental prognostic informations. The combination of PH and RV dysfunction is particularly ominous. Thus, the estimation of PASP should be considered in the standard assessment of ambulatory HF patients and we must study it as a therapeutic way in this population

## 0370

### Long-term cardiac prognosis and risk stratification in 260 adults presenting with mitochondrial diseases

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**Background:** The long-term cardiac prognosis of adults with mitochondrial diseases is unknown.

**Methods and Results:** Between January 2000 and May 2014, we retrospectively included in this study 260 consecutive patients (60% women)  $\geq 18$  years of age, [interquartile range (IQR) = 31 to 54], with genetically proven mitochondrial diseases, including 109 with mtDNA single large-scale deletions, 64 with the m.3243A>G mutation in MT-TL1, 51 with other